CLAIMS

1. A method for inhibiting lectin complement pathway (LCP) associated complement activation, comprising administering an effective amount of a mannose binding lectin (MBL) inhibitor to inhibit LCP associated complement activation in a subject.

2. The method of claim 1, wherein the LCP associated complement activation mediates a cellular injury.

3. The method of claim 2, wherein the cellular injury mediated by LCP associated complement activation contributes to tissue injury associated with atherosclerosis.

4. The method of claim 2, wherein the cellular injury mediated by LCP associated complement activation contributes to tissue injury associated with the pulmonary system.

5. The method of claim 4, wherein the MBL inhibitor is administered to the subject by an aerosol route of delivery.

6. The method of claim 2, wherein the cellular injury mediated by LCP associated complement activation contributes to tissue injury associated with arthritis, myocardial infarction, ischemia, reperfusion, transplantation, cardiopulmonary bypass (CPB), stroke, acute respiratory distress syndrome (ARDS), systemic lupus erythematosus (SLE), lupus, or dialysis.

7. The method of claim 2, wherein the cellular injury mediated by LCP associated complement activation contributes to tissue injury associated with ischemia.

8. The method of claim 2, further comprising administering to the subject a therapeutic treatment for treating an MBL mediated disorder associated with the cellular injury mediated by LCP associated complement activation.

9. The method of claim 8, wherein the therapeutic treatment is a drug.

10. The method of claim 8, wherein the therapeutic treatment comprises revascularizing a coronary artery.

11. The method of claim 10, wherein the revascularizing of a coronary artery is achieved by a method comprising percutaneous transluminal coronary angioplasty.

12. The method of claim 1, wherein a mammalian cell with a surface exposed MBL ligand is contacted with the MBL inhibitor.

13. The method of claim 1, wherein the MBL inhibitor binds to MBL.

14. The method of claim 13, wherein the MBL inhibitor is an MBL binding peptide, protein, antibody, or antibody fragment.

15. The method of claim 13, wherein the MBL inhibitor binds to a human MBL epitope.

16. The method of claim 15, wherein the human MBL epitope is a region of MBL which interacts with a monoclonal antibody produced by: i) hybridoma$_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) hybridoma$_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) hybridoma$_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

17. The method of claim 13, wherein the MBL inhibitor competes for binding to MBL with a monoclonal antibody produced by: i) hybridoma$_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) hybridoma$_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) hybridoma$_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

18. The method of claim 13, wherein the MBL inhibitor comprises an MBL binding CDR3 region or functional variant thereof.

19. The method of claim 18, wherein the CDR3 region is of a monoclonal antibody produced by: i) hybridoma$_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) hybridoma$_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) hybridoma$_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

20. The method of claim 18, wherein the MBL inhibitor further comprises a CDR2 region or a functional variant thereof.

21. The method of claim 18, wherein the MBL inhibitor further comprises a CDR1 region or a functional variant thereof.

22. The method of claim 13, wherein the MBL inhibitor is an antibody fragment.

23. The method of claim 22, wherein the antibody fragment is an antibody fragment selected from the group consisting of an F(ab')$_2$ fragment, an Fd fragment, an Fv fragment, and an Fab fragment.

24. The method of claim 22, wherein the antibody fragment is of a monoclonal antibody produced by: i) hybridoma$_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) hybridoma$_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) hybridoma$_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

25. The method of claim 13, wherein the MBL inhibitor is an antibody.

26. The method of claim 25, wherein the antibody is a monoclonal antibody.

27. The method of claim 26, wherein the monoclonal antibody is produced by: i) hybridoma$_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) hybridoma$_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) hybridoma$_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

28. The method of claim 25, wherein the antibody is a single-chain antibody.

29. The method of claim 25, wherein the antibody is a humanized antibody.

30. The method of claim 1, wherein the MBL inhibitor inhibits C3b deposition.

31. The method of claim 30, wherein the MBL inhibitor is an MBL binding peptide, protein, antibody, or antibody fragment and inhibits C3b deposition with an EC50 of between 10^{-9} to 10^{-7} mol/L.

32. The method of claim 1, wherein the method further inhibits VCAM-1 expression.

33. The method of claim 1, wherein the MBL inhibitor binds to a mannose-binding lectin-associated serine protease (MASP) or mannan.

34. The method of claim 33, wherein the MBL inhibitor is a peptide, protein, antibody, or antibody fragment.

35. The method of claim 34, wherein the antibody or antibody fragment is a monoclonal antibody or monoclonal antibody fragment.

36. The method of claim 34, wherein the antibody or antibody fragment is humanized.

37. The method of claim 34, wherein the antibody or antibody fragment is a single-chain antibody.

38. The method of claim 33, wherein the MASP is MASP-1 or MASP-2.

39. The method of claim 1, wherein the method is a screening assay.

40. A method for inhibiting cellular injury in a subject, comprising administering an effective amount of an MBL inhibitor to inhibit cellular injury in the subject.

41. The method of claim 40, wherein the cellular injury contributes to tissue injury associated with atherosclerosis.

42. The method of claim 40, wherein the cellular injury contributes to tissue injury associated with the pulmonary system.

43. The method of claim 42, wherein the MBL inhibitor is administered to the subject by an aerosol route of delivery.

44. The method of claim 40, wherein the cellular injury contributes to tissue injury associated with arthritis, myocardial infarction, ischemia, reperfusion, transplantation, CPB, stroke, ARDS, SLE, lupus, or dialysis.

45. The method of claim 40, wherein the cellular injury contributes to tissue injury associated with ischemia.

46. The method of claim 40, further comprising administering to the subject a therapeutic treatment for treating an MBL mediated disorder associated with the cellular injury.

47. The method of claim 46, wherein the therapeutic treatment is a drug.

48. The method of claim 46, wherein the therapeutic treatment comprises revascularizing a coronary artery.

49. The method of claim 48, wherein the revascularizing of a coronary artery is achieved by a method comprising percutaneous transluminal coronary angioplasty.

50. The method of claim 40, wherein the MBL inhibitor inhibits MBL deposition on a mammalian cell with a surface exposed MBL ligand.

51. The method of claim 40 or 50, wherein the MBL inhibitor binds MBL, MASP or mannan.

52. The method of claim 51, wherein the MBL inhibitor is a peptide, protein, antibody, or antibody fragment.

53. The method of claim 52, wherein the antibody or antibody fragment is a monoclonal antibody or a monoclonal antibody fragment.

54. The method of claim 52, wherein the antibody or antibody fragment is humanized.

55. The method of claim 52, wherein the antibody or antibody fragment is a single-chain antibody.

56. The method of claim 51, wherein the MASP is MASP-1 or MASP-2.

57. The method of claim 51, wherein the MBL inhibitor binds to a human MBL epitope.

58. The method of claim 57, wherein the human MBL epitope is a region of MBL which interacts with a monoclonal antibody produced by: i) hybridoma$_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) hybridoma$_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) hybridoma$_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

59. The method of claim 52, wherein the MBL binding peptide, protein, antibody, or antibody fragment competes for binding to MBL with a monoclonal antibody produced by: i) hybridoma$_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) hybridoma$_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) hybridoma$_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

60. The method of claim 40, wherein the MBL inhibitor inhibits C3b deposition.

61. The method of claim 60, wherein the MBL inhibitor is an MBL binding peptide, protein, antibody, or antibody fragment and inhibits C3b deposition with an EC50 of between 10^{-9} to 10^{-7} mol/L.

62. The method of claim 40, wherein the method further inhibits VCAM-1 expression.

63. The method of claim 51, wherein the MBL inhibitor has an MBL binding CDR3 region or functional variant thereof.

64. The method of claim 63, wherein the CDR3 region is of a monoclonal antibody produced by: i) hybridoma$_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) hybridoma$_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) hybridoma$_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

65. The method of claim 63, wherein the MBL inhibitor further comprises a CDR2 region or a functional variant thereof.

66. The method of claim 63, wherein the MBL inhibitor further comprises a CDR1 region or a functional variant thereof.

67. The method of claim 40, wherein the MBL inhibitor is an antibody fragment.

68. The method of claim 67, wherein the antibody fragment is an antibody fragment selected from the group consisting of an $F(ab')_2$ fragment, an Fd fragment, an Fv fragment, and an Fab fragment.

69. The method of claim 68, wherein the antibody fragment is of a monoclonal antibody produced by: i) $hybridoma_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) $hybridoma_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) $hybridoma_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

70. The method of claim 40, wherein the MBL inhibitor is an antibody.

71. The method of claim 70, wherein the antibody is a monoclonal antibody.

72. The method of claim 71, wherein the monoclonal antibody is produced by: i) $hybridoma_{(3F8)}$ deposited under ATCC Accession No. HB-12621, ii) $hybridoma_{(2A9)}$ deposited under ATCC Accession No. HB-12620, or iii) $hybridoma_{(hMBL1.2)}$ deposited under ATCC Accession No. HB-12619.

73. The method of claim 70, wherein the antibody is a single-chain antibody.

74. The method of claim 70, wherein the antibody is a humanized antibody.

75. The method of claim 40, wherein the method is a screening assay.